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EFFECT OF ENVIRONMENTAL TEMPERATURE AND DIETARY ENERGY ON FEED INTAKE IN CHICKENS

by

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A DISSERTATION

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and Dr. T. E. Hartung

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REVIEW OF LITERATURE

INTRODUCTION

There has been considerable interest in the phenomenon of feed intake. Feed intake of animals used in agriculture is of great importance for economic reasons. No other factor has such an overriding influence on cost of production as feed. Directly or indirectly, feed intake is an important aspect of all animal research. In humans, food intake has great significance due to possible disease problems associated with it and also the related cosmetic and social values, particularly of obesity. The terminology, "control of feed intake" and "regulation of feed intake", has been used interchangeably in the past. Brobeck (1965) and Stevenson (1969) differentiated between control and regulation, and suggested that control be used when referring to feed intake, since there are several factors being controlled to result in a regulation of energy balance. The terminology, "short term" and "long term" control is also used regarding feed intake mechanisms. The mechanisms for controlling meal to meal feeding are "short term" control while the mechanisms for controlling feed intake over a period of weeks and months for body weight maintenance are "long term" control. The terms, satiety, appetite, hunger, reward and pleasure have been used extensively in the past but recently have been used only infrequently. Morgane (1969) pronounced, in his introductory remarks to the conference on Neural Regulation of Food and Water Intake, that the subjective terminology

had served to divert attention from many of the fundamental physiological issues at stake, without really adding information on the mechanisms involved. So, in the context of this review the usage of such terms will be limited only to the literary meanings rather than any complex definitions.

The control of feed intake can be discussed either by considering the factors involved in the control or the anatomical basis for the function of the factors. This review emphasizes the mechanisms by which the factors are thought to function in controlling feed intake. Since the mechanisms of feed intake control are not clearly understood, the major evidence for and against these is presented. The anatomical aspects, which are usually concerned with the "feeding and satiety centers", are discussed in the latter part of this review.

FACTORS CONTROLLING FEED INTAKE

Role of the thermostatic mechanism

Booth and Strang (1936) proposed the necessary inhibition of eating is caused by the associated heat, based on observations of normal and obese humans. The elevation of skin temperature which followed a meal was correlated with the onset of satiety. The sensation of warmth in the skin was considered to cause a feeling of comfort and satiation. Later, Brobeck (1948) stated that "animals eat to keep warm and stop eating to prevent hyperthermia". This statement has been severely criticized by many including Kennedy (1953), Mayer (1964), Grossman (1968) and Soulairac (1969). Defend-

ing the hypothesis in general, Brobeck (1960) continued to regard the interpretation as an interesting possibility.

Winchester and Morris (1956) studied feeding behavior of cattle from different parts of the world. High temperature reduced the feed intake of European as well as Indian cattle. Johnson et al. (1957) noted that male and female rabbits exposed to 48°F. (8.9°C.) consumed more feed than at 83°F. (28.3°C.). Similar results were reported for Shorthorn cattle, but not for Zebu. This may have been due to Zebu cattle being genetically more adaptive to hot environments than other cattle, however, the highest temperature to which the cattle were exposed was only 80°F. (26.7°C.). Appleman and Delouche (1958) changed the environmental temperature from 20 to 0°C. and 20 to 40°C. and found the average time spent by goats eating hay was decreased at the higher environmental temperature. The higher environmental temperature also resulted in an increase in rectal temperature. In pigs, Sugahara et al. (1970) noted a 21% increase in feed intake in a cold environment of 7°C. and a 32% decrease in a warm environment temperature. Wilson (1967) observed the maximum feed intake in growing chickens at 16°C. and the minimum at 32°C. in a study using 16, 21, 27 and 32°C. environmental temperatures. O'Neill et al. (1971) reported a decreased metabolizable energy requirement with increased environmental temperature for both defeathered and feathered cockerels. The results of Kendeigh (1949) showed almost a

linear increase of energy intake with decreasing environmental temperature in English sparrows.

In poultry, some researchers have quantitated the change in feed intake related to changes in the environmental temperature. The reduction in feed intake was estimated to be 1.6% per degree rise between 17 and 29°C. (Payne, 1966) and about 5% per degree rise between 27 and 38°C. (Wilson, 1949). Smith (1971) based the metabolizable energy intake of chickens on metabolic body size ($kg.^{0.75}$; Kleiber, 1961) and noted the feed intake reduction was curvilinear within the range 70 to 100°F. (21.1 to 37.8°C.). Smith and Oliver (1972) observed a curvilinear decrease in the metabolizable energy intake of pullets between 21 to 35°C. and predicted that feed intake would have ceased at approximately 42°C.

All studies have not supported the existence of a thermostatic mechanism. Shapiro and Consolazio (1959) and Consolazio and Shapiro (1964) found a significant increase in the energy requirements of soldiers living and working under extremely high temperatures. It was suggested this resulted from an increased requirement of energy due primarily to the increased heat load in hot environments. There were apparently certain limitations in the study. No attempt was made to keep the various temperatures constant everyday and the attitude of the men might have influenced the results. Mullick (1964), working with buffaloes and cattle, also noted an increased intake of gross energy and digested energy in summer as compared to winter. However,

the experiments were not performed in controlled environments.

An explanation for the function of the thermostatic mechanism was proposed by Brobeck (1960). He suggested the specific dynamic effect was involved. The phenomonon is more commonly known as specific dynamic action although it is a less appropriate term according to Kleiber (1961). Other terms used synonomously are heat increment, calorigenic effect, thermogenic effect and thermal energy. Specific dynamic effect has been defined as the extra heat the animal produces due to the assimilation of feed. The extra heat of the specific dynamic effect may act through thermoregulation to inhibit eating, since the heat must be dissipated to maintain body temperature.

Some research has indicated an involvement of the specific dynamic effect. Forbes et al. (1934) gave different amounts of feed to rats and noted greater heat production with greater feed consumption. Passmore and Ritche (1957) gave experimental meals to humans in starved and satiated conditions while measuring heat production. The energy expenditure started increasing immediately after the meals in both cases. It was concluded the rise in heat production, which reached a maximum in about 40 minutes, was the result of specific dynamic effect. Protein is considered to have the greatest specific dynamic effect of all the nutrients (Brody, 1945). Ahmedov (1966) used two levels of protein under high and low environmental temperatures in dogs. With a high protein diet and a high environmental temperature,

there was more heat production than with a low protein diet and a low environmental temperature.

While the hypothalamus was recognized as the body temperature regulating center (Strom, 1950), Anand and Brobeck (1951a) suggested the hypothalamus was a mediating center for control of feed intake. Then, Brobeck (1960) proposed that feed intake might be affected by the same stimuli that affects the body temperature regulating center.

Attempts have been made to investigate this proposal. Andersson and Larsson (1961) caused a satiated goat to eat by cooling the anterior hypothalamus and preoptic area. By warming the same areas, eating was inhibited in a fasted goat. The study has been widely criticized on the grounds that unphysiological temperature changes were used. Bhattacharya and Warner (1968), using a different experimental approach, noted an increase in feed intake and a decrease in tympanic temperature of heifers when cold water was infused into the rumen, and the converse.

On the other hand, some researchers have concluded no causal relationship between feed intake and hypothalamic temperature. Rampone and Shirasu (1964) reported an increase in brain temperature of rats regardless of the proportion of protein in the diet. It was interpreted that the increased brain temperature was the result of reduced removal of endogenous heat produced in the brain, since relatively more blood might have gone to the gastro-

intestinal region for assimilation of feed. Baile and Mayer (1968) observed either no change or a decrease in hypothalamic temperature after force feeding goats intraruminally. It was concluded that ingestion of feed did not cause an increase in hypothalamic temperature, however, no attempt was made to explain what would have caused a decrease in hypothalamic temperature. Grossman and Rechtschaffen (1967) gave feeds of various temperatures to cats and rats and concluded the increase in brain temperature during and after feeding was not due to feed, but to activity of the jaws and mouth associated with eating. Dinius et al. (1970), using goats, reached the same conclusion.

Most researchers who could not relate the hypothalamic temperature or the specific dynamic effect to feed intake, did not seem to recognize the role of the thermostatic mechanism. However, since most research has shown an inverse relationship between environmental temperature and feed intake, it does not seem justified to discard the thermostatic mechanism of feed intake control. It must be noted that the relationship of hypothalamic temperature or specific dynamic effect to the control of feed intake is questionable. There still is a need to clarify the relationship of the control of feed intake to thermoregulatory process, as proposed by Brobeck (1960). Most of the experiments conducted to resolve the relationship

have measured only one aspect of the physiological response at a time. No attempts have been made to observe changes in body temperatures while making heat production measurements, and the converse. Experiments in which small changes in the temperature of various parts of the body are measured simultaneously with precise heat loss and heat production determinations, would help resolve the issue.

Role of the chemostatic mechanism

It has been suggested that animals can sense the calories in their body and the statement has been made that animals "eat for calories" (Adolph, 1947). As the calorie is a unit of energy brought about by catabolism of chemical compounds, the term, chemostatic has been used in referring to the mechanism whereby animals adjust energy intake. This term has also been used to include carbohydrates, lipids and proteins. The use of the term, glucostatic, is questionable, since compounds in addition to glucose are involved in the mechanism. In ruminants, volatile fatty acids are considered to be the major compounds involved (Eldsen and Phillipson, 1948). Many studies, have shown that animals generally eat to meet their energy requirements (Scott et al., 1947; Scott, 1948; Hill et al., 1956; Berg and Bearse, 1956; Bolton, 1958; Peterson et al., 1960; Seberell, 1961; Thayer et al., 1965; Caligado and Quisenberry, 1967; Gleaves et al., 1968; Gleaves and Dewan, 1971; Smith, 1971; and Smith and Oliver, 1972). However, Grossman (1955)

stated the body does not make rapid and fine adjustments to increased energy loads by decreasing feed intake. His studies, though, were not all dependent on voluntary intake. Morris (1968), after reviewing many studies of laying chickens, concluded that the adjustment was not perfect and chickens fed high energy diets usually overconsumed energy.

Montgomery and Baumgardt (1965) noted that ruminants adjust feed intake to the physiological demand for energy, if rumen capacity did not limit consumption. Cowsert and Montgomery (1969) observed an increase of dry matter intake as the energy level of the feed was decreased in ruminants. Since differences in the physical forms of ruminant diets exist, Baumgardt (1970) proposed that density should be included in addition to energy when describing the nutritive value of diets. He coined the term, "caloric density", meaning digestible energy calculated per milliliter of feed rather than digestible energy per gram of feed.

The importance of blood glucose concentration in the control of feed intake was recognized early. Bulatao and Carlson (1924) noted gastric contractions in dogs made hypoglycemic and the contractionsstopped with hyperglycemia. Mayer (1953) increased blood glucose levels in rats by injections of glucose, fructose or epinephrine, whereupon the rats decreased feed intake. When blood glucose concentration was decreased by injections of insulin, the rats ate more. On the other hand, injections of sucrose or fat emulsions, which did not affect blood glucose levels, had

no influence on feed intake. Muto and Miyahara (1972) used high and low protein diets, with and without the availability of 40% sucrose and observed the energy intake of rats was not affected by the source of energy. It was concluded that young rats chose sucrose rather than protein even at the sacrifice of growth and health. Steffens (1969) reported extensive evidence correlating levels of blood sugar, insulin, and free fatty acids with initiation and cessation of feeding in unrestrained rats.

Manning et al. (1959) were the first to show that increased blood glucose concentrations had no effect on feed intake in ruminants. It was suggested that acetate might be involved in feed intake control in ruminants. Simkins et al. (1965) considered acetate and ketone compounds as the most probable stimuli for chemoreceptors in ruminants since the concentrations increased in blood after feeding.

Mayer (1953 and 1955) presented various types of evidence to explain the glucostatic mechanism of feed intake control. Vagotomy abolished the normal gastric response to hypoglycemia. Hyperglycemia produced in the isolated head of a dog, connected to the body only through the nerve supply, resulted in hypoglycemia of the body. In normal and diabetic animals, and in animals subjected to various hormonal treatments, decreases in glucose utilization correlated well with increased feed intake. There was a positive correlation between liver glycogen and feeding behavior. Glucose injections into the cerebral ventricles

also depressed feed intake (Herberg, 1960). Anand et al. (1961) noticed distinct changes in the electroencephalographic recordings of the hypothalamic centers during hunger and satiety and found the changes were correlated with the level of glucose utilization in the body. The glucostatic concept was strengthened by use of gold thioglucose to degenerate the ventromedial hypothalamus (Marshall et al., 1955). Permanent overeating and obesity resulted. It was explained that since glucose had a selective affinity for the cells, the gold thioglucose destroyed the ventromedial hypothalamus (satiety center) resulting in loss of control of eating.

After Grossman (1955) demonstrated in dogs and humans, that hyperglycemia did not necessarily produce a state of satiety, Van Itallie and Hashim (1960) suggested that hunger and satiety states were correlated with arteriovenous glucose differences rather than just levels of blood glucose. Additional evidence that glucose utilization, not glucose level, is the critical stimulus was obtained by injecting monkeys with an unmetabolizable glucose, 2-deoxy-D-glucose (Smith and Epstein, 1969). This resulted in increased feed intake in spite of high total sugar in the blood. The metabolic paradox was due to the glucoreceptors being blocked by 2=deoxy=D=glucose. On the contrary, Le Magnen (1971) concluded from the same research that the ventromedial glucoreceptors were not directly involved in feed intake

control.

Epstein (1960), Fisher and Coury (1962), and Wagner and De Groot (1963), did not see any change in the feeding behavior as a result of injection of glucose into the hypothalamic region. Grossman (1967) stated that even by injecting insulin into the region of both the medial and lateral hypothalamus, no changes in food or water intake were observed. However, no research is known where insulin was accompanied by glucose injections.

Warner et al. (1971) reported that in ruminants there was an adjustment in energy consumption within 15 minutes after initiation of feeding and that precise adjustment occurred within three hours. It seemed the study supported the explanation of Mayer (1953 and 1955) for the glucostatic mechanism, but it was concluded that some unidentified blood component was responsible for the control of feed intake in ruminants. Thye et al. (1970) stated the chemostatic mechanism probably involves a complex interaction of many factors, of which circulating levels of absorbed nutrients might only be a part. Contrary to the research with ruminants, Powell et al., (1972) reported it took nine days for hens to adjust feed intake based on energy intake.

Animals in general eat to meet their energy needs. Blood glucose utilization is the major phenomonon involved in monitoring energy for feed intake control. However, the relatively long time required to adjust to energy needs

points out that blood glucose utilization is not solely responsible for the chemostatic mechanism. In addition, volatile fatty acids, ketone bodies and some unidentified blood components have been suggested to play a major role in ruminants. As yet, it is unresolved whether the hypothalamus is the only site of chemoreceptors for blood components responsible for the chemostatic mechanism of feed intake control.

Role of the lipostatic mechanism

In 1953, Kennedy proposed a lipostatic mechanism for the long term control of feed intake. It was explained to function by sensitivity to the concentration of all circulating metabolites, including the metabolites developed as a result of changes in fat depots. The data of Bates et al. (1955) suggested the amount of endogenous fat mobilized daily in ad libitum feeding conditions is proportional, within each type of fat, to the size of the fat depots. Teitelbaum (1961) and Cohn and Joseph (1962) observed, with animals made obese by insulin treatment or by force feeding, that feed intake decreased after the treatment was stopped until the normal weight was attained. Lepkovsky and Furuta (1971) also found that cockerels stopped eating for 6-10 days after being made obese by force feeding. Hamilton (1972), in a seven-year study on monkeys, postulated that the long term control of feed intake was the result of regulated body weight.

In ruminants, Balch and Campling (1962) suggested the

amount of body fat may activate a feedback mechanism for long term control over feed intake. Also in support of the lipostatic mechanism, Bines et al. (1969) observed that thin cows ate about 31% more hay and 23% more concentrates when given with a small fixed amount of hay than did the same animals when fat. Critics have suggested that part of the difference in feed intake between thin and fat cows may be due to physical limitations caused by accumulation of abdominal fat. This suggestion has support from the data of Lepkovsky and Furuta (1971) in which about 100 times more abdominal fat was found in force fed cockerels than in controls.

Assuming that circulating blood metabolites may be a factor in control of feed intake, Bines et al. (1969) suggested the adipose tissue of thin animals was probably more active and might remove such inhibitory factors from the blood, thereby reducing the inhibitory effect on appetite. Another possibility suggested was that inhibitory metabolites do not affect a thin animal to the same degree as a fat animal. An earlier suggestion by Wertheimer and Shapiro (1948), that nervous pathways run between the hypothalamus and the fat depots by way of the sympathetic system, has been revised to only efferent regulation of stored fat (Shapiro and Wertheimer, 1956). The suggestion, therefore, cannot explain the function of the lipostatic mechanism.

Under normal conditions, the lipostatic mechanism

apparently is operative in the long term control of feed intake. It appears the concentrations of all circulating metabolites are involved in feed intake control. The questions of which metabolites are involved and how the metabolites function in the lipostatic mechanism are still unanswered.

Role of the gastrointestinal tract

The upper portions of the gastrointestinal tract have been suggested to influence the control of feed intake. Oral factors were observed to be involved in the control of feed intake, since in esophagostomized dogs the period of sham feeding ceased even though the stomach was empty (Janowitz and Grossman, 1949). It has been observed (Teitelbaum and Epstein, 1962) that the rat cannot recover from "lateral hypothalamic syndrome" if the oropharyngeal sensations are not operative. Borer and Epstein (1965) observed rats using bar pressing to cause oral and intragastric drinking. With various concentrations of salt and sucrose solutions the preferences for salty and sweet solutions were the function of taste when the rats were neither hungry nor thirsty. The preferences were generated by oropharyngeal sensations. Maller et al. (1967) even stated that the passage of food through the mouth serves functions beyond that of stimulating taste and smell receptors. By tying off the esophagus and introducing solutions of labeled compounds, considerable radioactivity was observed in the brain but little in the

blood or liver. However, if the isotopes were introduced into the gut, radioactivity was not detectable in the brain but was demonstrated in the liver and blood. Oropharyngeal sensation was reported to be responsible for choice of feed and a motivator of taste and smell, but was not considered to be essential for the normal operation of feed intake control (Epstein, 1967).

Early in the present century, Cannon and Washburn (1912) reported that deprivation of feed and the subjective feelings of hunger-pangs were accompanied by rythmic contractions of the stomach. Quigley et al. (1929) stated that each peristaltic wave over the stomach produced one hunger pang. Janowitz and Grossman (1949) concluded that duration of eating is related to the size of intragastric feeding, and inert bulk in the stomach was as effective as feed in producing short term inhibition of eating. In experiments by Janowitz and Hollander (1955), the effect of intragastric feeding of 50, 100 and 175% of oral intake was studied, in dogs for many weeks. Adjustments did occur in the expected directions, but required weeks to become established and were never completely compensatory. The control system, therefore, was considered to be active but had a high degree of inertia. In pigeons, Cardini (1971) observed that intracrop preloading of less than 60% resulted in a directly proportionate decrease in oral intake. With more than 60% intracrop preloading, the decreases in oral intake were